Ventricular haemodynamic parameters were also measured, include HR, LVSP. Left ventricular myocardial was separated and cut to five slice. After experiment, the myocardial was used for myocardial infarction size evaluated with TTC stained. Immunohistochemical staining for Phosphorylation Akt and GSK-3β expression.

Results Ischemic postconditioning reduced LDH, CK and improved the haemodynamic parameters, and reduced myocardial infarction size (29.5% vs 47.3%). phospho-Akt and phospho-GSK-3β expression increased markedly in IPost group. Wortmannin may reduced phospho-Akt expression, and phospho-GSK-3β expression increased in I/R+SB group.

Conclusion Ischemic postconditioning may synergically protect myocardium in isolated rat heart. Wortmannin, a inhibitor of Akt, may weaken the cardioprotection effect of postconditioning. SB216763, as a inhibitor of GSK-3β, can simulate cardioprotection effect of postconditioning. Akt and GSK-3β play important role in the mechanism of signal pathway in ischaemia postconditioning.

e0120 THE IMPACT OF DIABETES ON THE ROLE OF REPERFUSION INJURY SALVAGE KINASE PATHWAY

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Objective To elucidate the effects of postconditioning on ischaemia/reperfusion cardiac and the role of reperfusion injury salvage kinase pathway in type 2 diabetic rats.

Methods The type 2 diabetic rats were induced by the intravenous injection of streptozotocin and high caloric diet. 60 Wister rats were divided into three groups randomly. Ischaemia- reperfusion in nomal rats (A group), ischaemia postconditioning in nomal rats (B group), ischaemia postconditioning in diabetic rats (C group). Rats were used for Langendorff isolated heart perfusion with 30 min of globe ischaemia and 60 min of reperfusion, then the models of Ischaemia-reperfusion (A) were made. But to B and C, rat hearts were subjected to six cycles of 10 min of globe ischaemia and 10 min of reperfusion as ischaemia postconditioning during the early minutes of reperfusion. Phosphorylation of akt and gsk-3β were analysed by western blotting and immunohistochemical staining.

Results phospho-akt and phospho-gsk-3β expression increased markedly in B group. But compared A group there were no parently diffrence in C group. phospho-akt and phospho-gsk-3β expression in C group is more less than in B group.

Conclusion Ischemic postconditioning may significiently protect myocardium in isolated nomal rat hearts. But in diabetic rats the protection of Ischaemic postconditioning has no effect, the mechanism of this phenomina maybe connected with gsk-3β in the condition of diabetic.

e0121 THE EFFECTS OF ENDOTHEIN-1 AND BQ-123 ON ATPASE ACTIVITY AND MRNA EXPRESSION IN AORTIC SMOOTH MUSCLE CELLS FROM SPONTANOUSLY HYPERTENSIVE RATS

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Aim To study the effects of endothelin-1 (ET-1) and BQ-123 (ET_A receptor antagonist) on activities and mRNA expression of ATPase in aortic smooth muscle cells (ASMCs) from spontaneously hypertensive rats (SHR) and Wistar-Kyoto (WKY) rats.

Methods The ASMCs were isolated from SHR and WKY rats. The ATPase activities of cultured ASMCs were determined by spectrophotography. The mRNA levels of Na^+, K^+-ATPase α_1-subunit and plasma membrane Ca^{2+}-ATPase isoform 1 ($PMCA_1$) were measured by semiquantitative reverse transcription PCR (RT-PCR).

Results 3 different concentrations of ET-1 (1×10^{-9}, 1×10^{-8} and 1×10^{-7} mol/l) significantly attenuated the activities of Na^+, K^+-ATPase and Ca^{2+}-ATPase and $PMCA_1$ mRNA expression (all $p<0.01$) in ASMCs from SHR. Three different concentrations of BQ-123 (1×10^{-8}, 1×10^{-7} and 1×10^{-6} mol/l) obviously prevented ET-1 mediated the inhibition of two kinds ATPase activities (all $p<0.01$) and downregulation of $PMCA_1$ mRNA expression ($p<0.01$). But the mRNA expression level of Na^+, K^+-ATPase α_1-subunit had no alteration after intervened by ET-1 ($p>0.05$).

Conclusions ET-1 may suppress Na^+, K^+-ATPase, Ca^{2+}-ATPase activities via ET_A receptor. The influence of ET-1 on Ca^{2+}-ATPase activity may partially occur in the transcriptional level. BQ-123 can inhibit the effect of ET-1 on two kinds ATPase activities of ASMCs in SHR by blocking the ET_A receptor.

e0122 EFFECTS OF CARDIOTROPHIN-1 C-TERMINAL PEPTIDES ON CARDIOMYOCYTE APOPTOSIS IN SD RATS FOLLOWING MYOCARDIAL ISCHAEMIA REPERFUSION INJURY

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Objective Observation of CT-1 C-terminal peptides in ischaemia -reperfusion injury before and after the intervention on myocardial cell apoptosis in SD rats.

Methods With ligation-release SD rats left posterior descending branch of coronary artery the ischaemia reperfusion heart model was established. 27 SD rats were randomly divided into four groups: Normal group (N, n=5); Disease group (D, n=6), Beginning of reperfusion after 30 min of MI; MI/R post-intervention group (T, n=8), Intraperitoneal injection of CT-1 C-terminal peptide (100 μg/kg) at same time of beginning of reperfusion after 30 min of MI; MI/R pre-intervention group (O, n=8), MI/R experiments was performed after intraperitoneal injection of CT-1C-terminal peptide (100 μg/kg) for 7 days. In accordance with the ECG monitoring results ended the experiment in animals dying, left the serum for examination of concentrations of CK and MDA and cut the ischaemic heart tissue and surrounding areas fixed in neutral solution of formaldehyde, paraffin-embedded and sliced. Using end-labelling TUNEL assay apoptosis of myocardial cells and calculate the cardiac myocyte apoptotic index (AI).

Results After MI/R, the average survival time of the disease group of SD rats was 93.17±24.7 min, that of MI/R pre-intervention group was 87.88±18.3 min. The average survival time of MI/R post-intervention group was 155.5±80.13 min, significantly longer than that of the disease and MI/R pre-(chronic) intervention group ($p<0.01$); The serum CK activity and MDA content and the myocardial apoptotic index (AI) around infarct area were increased significantly in disease group (N vs D, $p<0.01$), and which has been reduced significantly in the post-intervention group (T vs D, $p<0.01$). But still higher than that of normal group (q values were 5.197, 5.782, 7.391, respectively; $p<0.01$); The serum CK activity and MDA content were more higher in pre-ischaemia group than that in the disease group, and their apoptosis index (AI) is higher than that of normal group ($p<0.01$), but was no significant difference compared with the disease group ($p>0.05$); There were significant correlations between the myocardial apoptosis with myocardial injury and the extent of oxidative damage (r values were 0.9245, 0.8679, respectively; $p<0.01$).